

University of New Orleans

**ScholarWorks@UNO**

---

Department of Economics and Finance Working  
Papers, 1991-2006

Department of Economics and Finance

---

1999

## Homosexuality, birth order, and evolution: towards a equilibrium reproductive economics of homosexuality;

Edward M. Miller

*University of New Orleans*

Follow this and additional works at: [https://scholarworks.uno.edu/econ\\_wp](https://scholarworks.uno.edu/econ_wp)

---

### Recommended Citation

Miller, Edward M., "Homosexuality, birth order, and evolution: towards a equilibrium reproductive economics of homosexuality;" (1999). *Department of Economics and Finance Working Papers, 1991-2006*. Paper 19.

[https://scholarworks.uno.edu/econ\\_wp/19](https://scholarworks.uno.edu/econ_wp/19)

This Working Paper is brought to you for free and open access by the Department of Economics and Finance at ScholarWorks@UNO. It has been accepted for inclusion in Department of Economics and Finance Working Papers, 1991-2006 by an authorized administrator of ScholarWorks@UNO. For more information, please contact [scholarworks@uno.edu](mailto:scholarworks@uno.edu).

To appear Archives of Sexual Behavior (No. 29 (February 2000) No. 5)

Homosexuality, Birth Order, and Evolution:  
Towards a Equilibrium Reproductive Economics of Homosexuality

by

Edward M. Miller, PhD

Department of Economics and Finance

University of New Orleans

New Orleans, La. 70148

504-280-6913 (work)

504-280-6397 (fax)

504-283-3536 (home)

emmef@uno.edu

February 23, 1999

**Homosexuality, Birth Order, and Evolution:  
Towards a Equilibrium Reproductive Economics of Homosexuality**

**Abstract**

The survival of a human predisposition for homosexuality can be explained by sexual orientation being a polygenetic trait that is influenced by a number of genes. During development these shift male brain development in the female direction. Inheritance of several such alleles produces homosexuality. Single alleles make for greater sensitivity, empathy, tendermindedness, and kindness. These traits make heterosexual carriers of the genes better fathers and more attractive mates. There is a balanced polymorphism in which the feminizing effect of these alleles in heterosexuals offsets the adverse effects (on reproductive success) of these alleles contribution to homosexuality. A similar effect probably occurs for genes that can produce lesbianism in females.

The whole system survives because it serves to provide a high degree of variability among the personalities of offspring, providing the genotype with diversification, and reducing competition among offspring for the same niches. An allele with a large effect can survive in these circumstances in males, but it is less likely to survive in females.

The birth order effect on homosexuality is probably a byproduct of a biological mechanism that shifts personalities more in the feminine direction in the later born sons, reducing the probability of these sons engaging in unproductive competition with each other.

Running Title:

Keywords: Homosexuality, birth order, evolution, genetics

## Table of Contents

Introduction .....	1
Theoretical Explanations .....	2
Illustrative Example .....	6
Why Excessive Masculinity .....	7
Female Mate Choice .....	9
The Feminization of Homosexuals.....	9
The Feminization of Homosexual's Brains .....	10
Family Implications .....	14
Lesbianism .....	14
The Value of Male Variability .....	15
Frequency of Homosexuality versus Lesbianism .....	17
Alternative Theories for the Survival of Homosexuality .....	22
Number of Older Brothers and Homosexuality .....	22
Birth Order Effects on the Masculinity-Femininity Continuum .....	23
Adult Behavior and Birth Order .....	25
Socialization and Birth Order .....	26
Diversification by Birth Order .....	27
Mechanisms for the Birth Order Effects.....	27
Conclusion.....	31
References .....	32

## Introduction

Homosexuality is common. Its very commonness is a major evolutionary puzzle. How could genes for homosexuality have survived? Homosexuals, lacking attraction to females, produce relatively few descendants. Thus one would expect that whatever a population's initial homosexuality rate was, that rate would soon diminish to virtually zero. Yet appreciable homosexuality persists. How can this be?

Moran (1972), after reporting a study in which 95 homosexuals over 40 (i.e. after end of child bearing) had only 37 children (at least two offspring per male are needed for a stable population) concluded "if male homosexuality is a biological phenomenon, it does not seem possible for it to have a genetic basis." Hamer & Copeland (1994, p. 183) in their chapter on the evolution of homosexuality (the problem of this paper) report that "the openly gay young to middle-aged men we interviewed had approximately one tenth as many children as their heterosexual brothers," although noting that the earlier Bell & Weinberg (1978) study found that the gay men had one fifth as many children as their heterosexual reference group.

Another study found that of men over fifty (i. e. who should have completed their families) who had had sex with another man within five years (which included many bisexuals, and even a few heterosexuals, as well as homosexuals), most had been married (62.9%) and 56.4% had had children (Van de Ven, Rodden, Crawford, Kippax, 1997). While there was no control group, these figures are clearly less than would be found for a random sample of Australian men aged over 50 years in age. Rogers and Turner (1991) in their survey of studies found that homosexuals were less likely to be married. This paper aims to show how such low level of reproductive success could be consistent with homosexuality having a genetic basis.

This is not the place to go into great detail on the evidence for a genetic role in homosexuality (surveys are in McKnight, 1997; and Pillard and Bailey, 1998), but family and twin studies (Bailey and Martin, 1995; Bailey and Pillard, 1991; Bailey, et al. 1993) make it clear that there is an important genetic component. Pillard and Weinrich (1986) found evidence for the familial nature of male homosexuality by showing that brothers of male homosexuals were four times as likely to be homosexual as brothers of controls. Kallmann (1952) reported an incredible 100% concordance for homosexuality in monozygotic twins. Other studies, such as that by Whitam, Diamond, and Martin (1993) found much lower concordance rates. Bailey and Pillard (1991) found that monozygotic twins are concordant for homosexuality 52% of the time, and dizygotic twins only 22% of time, a statistically significant difference that points to a large role for genes.

A similar study of lesbians found 48% concordance for monozygotic twins and 16% for dizygotic twins, again suggesting an important genetic component (Bailey, et al. 1993). A later population based study found somewhat lower concordance rates (Bailey and Martin, 1995) but still pointed to substantial heritability. In the above Bailey et al. studies, the half or more of the monozygotic twins who were discordant in spite of identical genes and similar rearing, suggests that some environmental factor (which may be prenatal, or essentially random, as in Miller, 1997a) plays a role.

There is a large literature trying to explain how genes for homosexuality could have survived (most recently summarized in McKnight, 1997). Some (Wilson, 1975; Weinrich, 1976) have argued that homosexuality has such a strong positive effect on the reproduction of siblings that genes coding only for homosexuality could survive. However, an extremely strong positive effect on the siblings' reproductive success would be required for such a gene to survive, and any such effect would be very apparent (it is not). Also, as Weinrich (1987), LeVay (1993, pp. 128-129) and others have pointed out, if individuals are to devote their efforts to helping siblings, it would seem more productive for them to be asexual than to expend efforts pursuing members of their own sex (and being exposed to the risk of venereal disease that sexual activity brings).

It might be noted that the interest in the evolutionary survival of homosexuality is independent of whether specific genes linked to homosexuality exist. There is evidence for such genes (Hamer et al. 1993, Hamer & Copeland, 1994, Hu et al. 1995). However, even if none of the variability in sexual orientation was genetic, there would still be the interesting question of how did humans come to be genetically constituted so that a relatively large proportion (say 2%) were homosexual. Thus, to ask the question of why there are so many homosexuals does not require accepting that there is a "homosexual gene".

Suppose an "environmental" explanation for homosexuality (such as the stress hypothesis) comes to be accepted. There will still be the evolutionary question of how did humans evolve so as to have a genotype in which environmental factors could cause certain individuals to have a phenotype so poorly adapted to continuing the genotype as homosexuals are.

### Theoretical Explanations

It is fairly easy to imagine how asexuality could occur (the complicated mechanisms needed to recognize potential mates and be attracted to them need merely have a critical link broken by a mutation). Since there may be a certain irreducible number of errors in the sexual attraction process, a certain amount of asexuality could easily be explained.

However, attraction to the same sex is harder to imagine evolving from scratch. Yet since homosexuals do find themselves attracted to males, but not to females, it follows that brains do somehow reliably separate males from females. Such a finely tuned mechanism is highly unlikely to have withstood the rigors of natural selection unless it served a purpose. Most likely, the mechanism for attraction to males is present in the human genotype, but it is normally turned on only in females and in homosexuals.

The obvious purpose for such a brain mechanism is to cause mature females to be attracted to mature males, and ultimately to produce offspring. Since sex in humans is determined by the presence or absence of a Y chromosome, and this chromosome is very small (i.e. has limited capacity for carrying information), it is very likely that the information that permits a homosexual or another person to determine whether a person is male or female is carried on other than the Y chromosome. Since it is hard to imagine a mechanism emerging just so homosexuals can recognize and be attracted to other males, it is very likely that homosexuality involves the activation of this mechanism in the few males that become homosexuals, when it would normally be inactivated in males.

The general theory of sexual development is that it is controlled by hormonal signals (or the absence of such signals) and that once the signal is given it affects a wide range of development conditions, from the genitals to the brain (see Gandelman, 1992 or Ellis & Ames, 1987 for good summaries). Indeed, the prevailing wisdom is that humans are programmed to develop as females (which presumably includes a sexual attraction to males), and that testosterone from the fetal testes masculinizes the males, leaving the remainder to develop as females.

Since sexual orientation is just one of a number of traits that normally separate females from males, it is very likely that all or most of the sex specific traits are activated (or turned off) by a single hormonal mechanism, which occasionally is partially activated in males, producing homosexuality. Other traits (including personality ones) in which females differ from males, presumably are partially activated by the same signal that produces homosexuality. As will be argued, these feminizing influences frequently contribute to reproductive success in heterosexual males, and thus are enabled to perpetuate themselves.

For an allele (a variation of a gene) which frequently produces an effect in some individuals (homosexuals here) that is opposed to reproductive success to survive over the long run,<sup>1</sup> the allele must also contribute to reproductive success when in other individuals (which in this case are heterosexuals). Presumably two alleles at the same location produce

homosexuality, but one allele produces a desirable effect in heterosexuals. Versions of this theory have been discussed by McKnight (1997, elaborating on MacIntyre & Estep, 1993).

Another (and less discussed possibility) is for there to be pleiotropic genes that affect sexual orientation. A pleiotropic gene is simply one that has more than a single effect. One effect could be contributing to homosexuality (which has a negative effect on reproductive success). The other effect or effects could be to contribute to reproductive success in heterosexuals in some manner.<sup>2</sup>

There has been extensive research into the genetics of personality (see for instance Eaves, Eysenck, & Martin, 1989; Plomin, Chipuer, & Neiderhiser, 1994; Rowe, 1994). Consistently the models that give the best fit involve an additive genetic variance along with non-familial environmental effects, and sometimes familial effects. Sexual orientation can be viewed as just another aspect of personality, although a very important aspect. As is discussed in detail later, sexual orientation correlates with femininity on measures of personality traits related to masculinity-femininity. Thus it seems very plausible to use a polygene additive model for homosexuality.

Given that a beneficial effect on heterosexual's reproductive success is the most plausible (I would argue the only plausible) mechanism by which genes contributing to homosexuality could have survived, the question is how do these genes contribute to heterosexual's reproductive success?

How do we answer that question? These genes should produce the same effects in heterosexuals as they do in homosexuals. Thus, it is necessary to look at the traits homosexuals exhibit to a greater extent than heterosexuals (only some of which would carry any particular allele that contributes to homosexuality).

It is also necessary that the traits in question increase the reproductive success of the heterosexuals in which they will reside for most of the time. Thus, theory suggests that the genes of interest would contribute to homosexuality while also increasing heterosexual reproductive success.

A plausible possibility for the heterosexual reproduction increasing effects of a pleiotropic homosexuality producing gene would be found in the feminine traits of sensitivity, kindness, empathy etc. that are frequently exhibited by homosexuals. These traits would often make for a better father (see discussion later).

They may be more risk adverse and avoid the costs of inter-male contests (this point was suggested by Mike Waller). Werner (1998) has made this point, suggesting a simple system where two "homosexual" alleles made for a homosexual, and none for a dominant male who died early in fighting, while one with a single allele of the homosexual type



would be most successful. Amusingly, he ended his essay with Christ's comment "Blessed are the meek, for they shall inherit the earth."

It is easy to imagine that in most males these traits help attract females, and hence lead to greater reproductive success. Indeed, one reason why they might be attractive to potential mates would be that they contribute to being good fathers and good providers. The typical male without homosexuality related genes may be too masculine for optimal reproductive success (how this could come to happen will be discussed later), and greater femininity on a number of dimensions could contribute to his reproductive success.

Now imagine there are a number of alleles that make for femininity. If the typical male inherits only a few of the alleles that make for femininity, his reproductive success is increased. Only if he inherits a large number of such alleles is his development pushed so far in the "feminine" direction that the brain circuits for mate choice and sexual orientation produce homosexuality. Such genes could survive because most of these time they would be in heterosexual males and be contributing to reproductive success. Only occasionally would enough of these come together (probably along with unknown environmental factors) to produce homosexuality. Such alleles will be in a balanced polymorphism such that if they got any rarer, their contribution to reproductive success would be increased through making heterosexuals more successful. If they got much more common they would more often contribute to homosexuality and be selected against.

Although the above discussion is purposively a little vague on the mechanism, given the evidence for prenatal hormones playing a key role in sexual differentiation, it is expected that the genes that affect sexual orientation would have their effect through prenatal hormonal effects on sexual differentiation (Ellis & Ames, 1987). This same prenatal hormonal system seems also to affect sex typical behaviors of various types. For instance, that the behavior of human female twins who shared the womb with a brother is more masculine than that of those who shared it with a female suggests a hormonal effect on human behavior (Miller 1994, 1997b).

It is very plausible that there are pleiotropic genes, one of whose effect is to feminize the parts of the brain that control sexual orientation and another of whose effects is to feminize brain areas that affect personality, choice of childhood play objects, or choice of role model. Such genes might change the level of hormones during prenatal critical periods, or the receptor density, or the level of enzymes that convert one steroid to another, or the level of binding proteins, or the permeability of the blood-brain barrier to hormones, etc. Just enumerating some of the possible pathways makes it plausible that multiple genes could be involved, along with several environmental factors. One of the prenatal factors could be the

prenatal stress that has been argued to contribute to homosexuality (Dorner, Rohde, Stahl et al. 1975; Dorner, Schenck, Shmiedel, & Ahrens 1983; Ellis, 1987; Ellis, Ames, Peckham, & Burke, 1987).

**<Insert Diagram 1 about here>**

Diagram 1 illustrates the theory. The hormonal environment varies on a continuum from that which produces masculinity to that which produces femininity in brain organization. Those males who are subject to the most femininity producing environments become homosexuals. Those who are less extreme exhibit varying degrees of bisexuality.

### Illustrative Example

To illustrate the effect imagine that there are five different genes that play a role in locating a male along a homosexual-hyper-masculine continuum. Each gene has two alleles, one that moves the individual one step towards the feminine side, and one that moves it towards the masculine side. For simplicity, imagine each gene has the same effect. A male becomes homosexual only if he inherits all 5 homosexuals producing alleles. Computation shows that there is a  $1$  in  $2^5$ , or  $1/32$  chance of this happening. Thus, slightly more than 3% of the males will be homosexual. Males who inherit fewer than five such alleles are heterosexual. If they inherit any such alleles they benefit from an increase in sensitivity. Ones who inherit none of these alleles are hyper-masculine and lacking in the sensitivity, kindness, empathy, and other traits that make them most attractive to females.

In this simple example all genes are identical in effects so we can analyze any one gene. There are 16 different combinations of the alleles of the other four genes. Capitals are the alleles that make for homosexuality (in this case these genes are dominant, but that is not critical to the argument), and the genes are a, b, c, d, and f. Consider the F allele of the f gene. It will produce homosexuality only if it is inherited by an individual with the ABCD genotype. Only 1 out of 16 individuals will have this. If it is inherited by individuals with 1, 2, 3 or 4 of the femininity alleles, a, b, c, or d, it makes him more sensitive. This increases his reproductive success. These individuals are the other 15/16's of the population. In this set of circumstances, the gene will make a contribution to the reproductive success of the majority of the males and could easily survive. In this example, if the homosexuals have no descendants, (success of 0) and the other individuals receiving the allele have their reproduction increased to  $16/15$  or  $1.066$ , the allele will be reproduced in the next population, permitting the allele to maintain its frequency.

If the frequency of the homosexual allele F increases above .5 (and half of the other genes have the homosexuality producing alleles), in the next generation the reproductive success of the carriers of the F allele will be less than one. Thus

the F allele will tend to decrease in frequency. If the F allele is found with a frequency of less than .5, the reproductive success of the carriers will be greater than one, and the gene will tend to increase in frequency. Thus, the frequency of the F allele would remain at .5.

The argument is symmetrical, and exactly the same for every other gene. The above example is obviously very simple, but it does indicate how there could be a stable polymorphism with a number of genes contributing to homosexuality. Thus we should not reject the idea of a genetic role in homosexuality just because at first glance survival of a gene for homosexuality seems implausible.

Obviously such a model can be elaborated. For instance, it is quite possible that some males are already on the feminine side, so an additional dose of femininity is mildly adverse to their reproductive interests, while others are so masculine that there are greater than average benefits from being shifted in the feminine direction. But these are just complications, not essential alterations.

One or more of the factors in the example above could be environmental ones, or possibly a random factor. Monozygous twins frequently grow up discordant for sexual orientation in spite of identical genetic endowments and being raised in the same family in very similar manners (citations above). Judging from this fact, it is very likely that one element is either random, or depends on some relatively minor environment factor (which acts almost as random), such as position in the womb. Thus, there is nothing in the above idea that is inconsistent with one or more environmental factors, including prenatal stress, playing a major role in the etiology of homosexuality.

How large an effect is needed to sustain homosexuality? MacIntyre & Estep (1993) provide some calculations for simple genetic systems (not the one discussed above) and conclude a 2% fertility advantage in the heterosexuals would be capable of insuring the survival of genes for homosexuality. They point out that this is such a small number that it would probably be lost in the noise of most studies, and hence would not be detected.

### Why Excessive Masculinity

The close reader of the above argument may have noted that it started with a presumption that the typical heterosexual was overly masculine (i.e. overly masculine for maximal reproductive success). In turn, this implies that genes for certain feminine traits would increase the typical heterosexual's reproductive success. While one can certainly imagine males being so high in stereotypical masculine traits that they were unattractive to women (such males might be very ruthless, selfish, insensitive, cruel etc.) someone might object that evolution might not produce many such males. Would

this destroy the above argument? How can we be sure that in the absence of the genes contributing to homosexuality that such excessive masculinity would exist?

Actually, if excessive genetic femininity exists and produces homosexuality, it follows that in equilibrium the typical heterosexual male will be excessively masculine. Imagine that the heterosexual males were of just the optimal position on the masculine-feminine continuum for maximal reproductive success. However, if this were really the case, averaging in the effect of the induced homosexuality must imply an above equilibrium frequency of the femininity producing alleles. These alleles would be decreasing in frequency. The situation would not be in equilibrium. Why would this be so?

Every so often, the allele would contribute to homosexuality, and this would happen with a finite probability. With the homosexual reproductive success well below unity, and the reproductive success of the heterosexual carrier of the allele at its maximum (by the above assumption), the appropriately weighted average reproductive success of all the allele's carriers would be below the maximum, while the maximum will normally be above one. The alleles that contributed to homosexuality would have a lower reproductive success than those that moved these males towards hyper-masculinity. The hyper-masculinity producing alleles would be increasing in frequency.

This increase in frequency would only stop when the shift to hyper-masculinity had become sufficiently great that the benefits from tempering hyper-masculinity were great enough to offset the loss of reproductive success from homosexuality, and carriers of both alleles had the same reproductive success. Thus the requisite (for the above theory) excessive masculinity can be presumed to exist if there are alleles that both contribute to homosexuality and femininize excessively masculine males.

Actually, the disadvantage to having a high proportion of the genes that make for homosexuality includes not only homosexuality, but also being avoided for mating purposes by females. Why would females avoid the carriers of such genes? Because, with genetic reshuffling in the next generation, their descendents would have an above average risk of homosexuality. For instance, in the above simple example, a female who mated with a male with five of the six homosexual related genes would have an above average chance of having her son prove to be homosexual, an event that would reduce the number of grandchildren she would have. From her viewpoint the ideal male might have, say, three of the alleles that made for homosexuality. Such a male might be "feminine" enough to make a good father and provider, but yet be masculine enough to signal that he had few genes that made his offspring likely to be homosexual.

This does not change the basic argument of the model because the stronger the disadvantage to being on the feminine side of the distribution, the more selection will lead to the male being on the hyper-masculine side, where only the genes for femininity can temper his hyper-masculinity.

A more interesting possibility is that females may have evolved so as to adjust their choice of mate to the genes their own body is carrying. If the genes they carry would make for an appreciable risk of homosexuality in their sons, they should choose a mate who is on the strongly masculine side, thus minimizing the risk of homosexuality. At the other extreme, if the women carry genes for masculine traits, they can mate with men carrying more of the feminine traits without an exceptionally high risk of homosexuality in their sons.

### Female Mate Choice

When human females are asked to list the traits that are desirable in a mate, kindness is consistently listed of one of the most desirable (Buss, 1989, 1994; Buss & Barnes, 1989) in a study that collected data not just for Anglo-Saxon cultures, but for 37 different cultures. Sprecher (1989) documented that both sexes prefer more expressive partners. Later, Sprecher, Sullivan, & Hartfield (1994) reported that "women have expressed a greater preference than men for such personality characteristics as expressiveness, kindness, and considerateness." These are traditionally considered feminine traits (McKnight 1997), and it is plausible that one developmental mechanism could select for them and for a female typical choice of sexual partner (i.e. for a preference for male sexual partners).

It should be noted that there are good biological reasons for a female to seek a mate who is kind and considerate. He is more likely to give her resources for raising her offspring and not to physically harm her, or to emotionally harm her by making her jealous. The disadvantage to the male of being kind, considerate, empathetic, etc. may be that he devotes less time and resources to seeking second mating opportunities. Very likely, the best level of femininity etc. for a male varies with time and location, making it optimal for parents to have children that vary in this trait, as in other traits (see the discussion above under the Value of Male Variability).

### The Feminization of Homosexuals

A testable implication of the above theory is that the brains, behavior, and perhaps even bodies, of homosexuals would be more feminine. On tests, either the homosexuals would exhibit the female pattern, or they would be shifted in the female direction.

The theory predicts furthermore that this feminizing effect would be observed only for those characteristics that develop at the same time, or are subject to the same developmental mechanism as sexual orientation. However, since at present we do not know which characteristics these are, this aspect of the theory cannot be directly tested.

As to the timing of such effects, Hall & Kimura (1994) reported that the fingerprints of homosexuals are more often leftward shifted than those of heterosexuals. They also state that the volvar pads form on the fingertips by the eighth week of conception and that the prints are fully formed by about the 16th week. The homosexual/heterosexual differentiation presumably occurs at least partially during this period. This observation dates hormonal effects that could also be affecting the brain and the personality.

There is a large literature which points to prenatal hormonal effects on male/female differences and homosexual-heterosexual differences. Let us start with the physical ones (see the papers in Ellis & Ebertz, 1997).

Schlegel (1966, as cited by Eysenck & Wilson, 1979, p. 38-39) reports a difference in the pelvic structure of homosexuals. In females the pelvis is more barrel shaped with a relatively broad outlet (to facilitate child bearing) while in males it is more funnel shaped (with the result that even though men are larger than women on average, their pelvic outlets measure much less than that for women). He reports that homosexual males tended to have feminine-type pelvises.

Furthermore, Schlegel claims that pelvic type correlates with personality.

Quoting from Eysenck & Wilson's summary (1979, p. 38-39), "Masculine type pelvises correlated with leadership, an active sexual role, dominance and preference for younger sex partners in men and women alike. Feminine type pelvis correlated with empathy, suggestibility and compliance, as well as preference for older sex partners. . . . Schlegel even studied cows (whose pelvic outlets are more easily observed than those of human subjects!) and found that cows with narrow outlets, i. e. funnel-shaped pelvises, tended to mount other cows and generally behave in a more masculine manner."

The theory that the genes that lead to homosexuality also lead to feminine behavior explains the finding that homosexuals frequently report gender atypical boyhood behavior. Bailey & Zucker (1995) found in a meta analysis of studies that there was a strong tendency for homosexuals to recall childhood gender atypical behavior. Green (1987) found that "sissy" boys often grow up to be homosexuals. Presumably constitutional behavioral differences persist from childhood in adulthood.

## The Feminization of Homosexual's Brains

Having looked at physical differences, now let us look at psychological differences. For many traits homosexuals appear to be more like females than typical males. Pillard (1991) lists 31 studies in which psychological tests measuring putatively sex-dimorphic-traits were administered. In only one case (two others were mixed) did the homosexual or lesbian group fail to have scores that differed from their own sex in the direction of the opposite sex. Those interested in the pre-1990 studies are referred to his paper for details. For homosexual males (the majority of the studies reported on) this implied that homosexuals were more feminine than the heterosexuals.

More recently, Chung & Harmon (1994) found that gay men had lower masculinity scores, but not femininity scores, on the Bem sex role inventory than did heterosexual males.

Weinrich (1987) reports some research done by him and Pillard (unfortunately not reported elsewhere) using the Strong-Campbell Vocational Interest Inventory. To be precise, for the jobs that were significantly preferred by heterosexual women according to the test's norms, the homosexual men on average reported higher levels of interest than the heterosexual men did. The trend was so strong that we could "predict 65%-70% of the time whether a given subject was homosexual or heterosexual, knowing solely his Strong-Campbell scores. For a psychological test-and one not designed with homosexual/heterosexual discernment in mind-this figure is remarkably high."

The finding that male-female vocational interests are different is usually explained by socialization, girls are brought up to feel certain occupations are more suitable for them, and boys are brought up to prefer other occupations. However, the same explanation seems unlikely to apply to homosexuals since they are normally brought up as males, and subjected to strong pressures to adopt male patterns of behavior, to play with male toys, and to act out male occupational roles.

Tuttle & Pillard (1991) using a measure of femininity (34 true-false questions that constituted the Femininity Scale of the Personal Values Abstract of the California Personality Inventory) found that homosexual males scored 19.00 versus 15.62 for heterosexual males, and 20.74 for heterosexual females (high scores are more feminine). This difference was significant at the one in ten thousand level. Interestingly, the homosexual females scored 19.19, which is only slightly more feminine than the homosexual males. The homosexual female versus heterosexual female difference was statistically significant, although to a much weaker degree ( $p < .041$ ) than for the homosexual/heterosexual difference in males. Also, Salais & Foscher (1995) found gays more altruistic.

Ellis, Hoffman, & Burke (1990) examined self reports of male homosexual behavior, and found that there was a negative relationship between most forms of violent or criminal behavior and homosexuality. This is consistent with the homosexual brains being less masculinized than heterosexual brains.

Others (Gladue, Beatty, Larson, & Staton, 1990) report a more feminine pattern of spatial ability among homosexuals. For instance, Hall & Kimura (1995) show how on a throwing accuracy task men do better than women, but homosexual men are closer to the female pattern than are heterosexual males.

McCormick & Witelson (1991) found that homosexual men do less well at certain spatial tests than heterosexual men, and women are known to do less well at these tasks than men. This is consistent with a shifting of the structure of the brain in homosexual males in the feminine direction.

However, Tuttle & Pillard (1991) found that homosexual males did not have an ability pattern atypical of other males. One possible explanation for their results differing from the other studies is that their homosexual subjects consistently did better than heterosexual males on all of the four tests used, a possible result of a recruitment bias arising from recruiting their homosexual subjects from readers of gay newspapers. This may have resulted in some restriction of range.

Another study that did not fit the pattern is Halpern & Crothers, (1997). They found homosexuals to be more masculine than heterosexual males.

It is a popular observation that many homosexuals display "feminine" mannerisms and voice quality. Guadio (1994) has documented that most observers can tell which speakers are homosexual without being told in advance.

Also, Blanchard & Bogaert (1996a) and Bogaert & Blanchard (1996), show that homosexuals have an earlier puberty onset than heterosexuals. Since female puberty is earlier than that of males, this represents a shift in the female direction. This is consistent with the homosexual brain being somewhat feminized.

Hamer and Copeland (1998, p. 99) in a "highly preliminary result" looked at the female relatives (mothers and daughters) who were either linked or unlinked for Xq28 (the location where a major gene for homosexuality is believed to be located), reporting "the women with the gay version of Xq28 . . . had begun puberty on average of six months earlier than the other mothers." This supports the role of this gene in causing homosexuality, as well as in feminization.



The hypothalamus is believed to be important in the timing and regulation of puberty. LeVay (1991) made a well publicized finding that in homosexuals the size of a sexually dimorphic nucleus in the anterior hypothalamus was that of females rather than that of males.

Other brain differences are known. Swaab and Hofman (1990, also the review in Swaab, Zhou, Fodor, & Jofman, 1997) found that the vasopressin part of suprachiasmatic nucleus in homosexual men was twice as large in homosexual men as in heterosexual ones. The suprachiasmatic nucleus is known to be involved in regulating the circadian (daily) rhythms. Interestingly male rats treated with the aromatase inhibitor ATD (which hinders the conversion of testosterone into estrogen with the brain) and made bisexual prefer female rats when tested in the late dark phase and male rats when tested in the early dark phase (Bakker and Slob, 1997). The same ATD-treated bisexual rats had an increased number of vasopressin-expressing neurons in the suprachiasmatic nucleus (Swaab, Slob, Houtsmuller, Brand, & Zhou, 1995), suggesting a prenatal link between sex hormones and the brain that somehow produces homosexuality..

Also, Allen & Gorski (1992) reported that the size of the anterior commissure was about the same size in gay men as in straight women. As LeVay (1993, p. 123) points out "the very fact that the anterior commissure is not involved in the regulation of sexual behavior makes it highly unlikely that the size differences result from differences in sexual behavior." This makes it more likely that there is some process shifting certain male brains in the feminine direction.

Alexander & Sufka (1993) did electroencephalographic measures on homosexuals, females, and straight males, measuring brain waves during baseline conditions and while the subjects performed experimental tasks. They found that homosexuals differed from "both male and female heterosexual groups during baseline recordings and different than heterosexual males during judgement for verbal and spatial stimuli, but not significantly different from heterosexual females." (p. 273). This is very consistent with other findings in which homosexual males resemble heterosexual females, and is consistent with a shift in the female direction during brain development.

McKnight (1997), citing a personal communication from Alexander, states that preliminary findings from a large study support these findings and that "these EEG patterns are dynamic and change with the degree of homosexuality reported by the individual."

Reite, Scheeder, Richardson, & Teale, (1995) did magnetoencephalographic measurements on homosexual and heterosexual men, focusing on the M100 location in the superior temporal gyri. Straight men showed significantly more

anterior reactions in the right hemisphere as compared to the left one. Gay men did not demonstrate significant differences between the right and left hemispheres. They resembled the female pattern more than the male pattern.

In a study of event-related potentials, Wegesin (1998) found that slow wave activity recorded during mental rotation was greater for heterosexual men than for heterosexual women and gay men.

Then there are the controversial reports that homosexual brains when challenged with estrogen respond with release of luteinizing hormone (Dorner 1975, Gladue, Green, & Hellman, 1984), a result some have not found (Gooren, 1986). Since normal female brains respond with such a surge, this could be one more piece of evidence that homosexual brains are modified in the female direction.

### Family Implications

A frequent finding of studies of families of homosexuals is that the father was much less dominant than is found in most heterosexual families. More often than normally the mother made the key decisions and ran the family. For instance, Bell, Weinberg, & Hammersmith (1981, p. 66) reported that fewer homosexuals than heterosexuals had reported that their fathers had dominated their mothers, and more homosexual men had said that their mothers dominated their fathers. This finding is traditionally interpreted as a characteristic of the structure of families that produce homosexuality. However, it could also be a reflection of the personality of the fathers. If the fathers of homosexuals are relatively feminine (as would be expected from sharing half of their genes with their homosexual sons) they might very well take less of a leadership role in the home and be less dominant.

### Lesbianism

This paper has primarily focused on male homosexuality. Much less is known about lesbianism. However, much of what is known indicates that lesbians are relatively masculine in their interest and personalities as compared with male homosexuals. For instance, Spence & Helmreich (1978) used their Personal Attitudes Questionnaire to show that male homosexuals scored significantly lower on masculinity and significantly higher on femininity, while lesbians were significantly higher on masculinity and lower on femininity. Interestingly, lesbians actually scored higher on the masculinity scale than did male homosexuals.

Perkins (1981) reported that lesbians had narrower hips and more muscular builds than non-lesbian women. This implies they were more like men. Within lesbians, those that played the more dominant (more male like role) were taller (dominants compared with passives gave a two inch difference and was statistically different). The dominants also had

broader shoulders and narrower hips than did lesbians who played passive or intermediate roles, although only the shoulder differences were statistically significant. These differences are consistent with one developmental process affecting both masculinity in build and in personality.

The most powerful evidence for lesbianism being caused by prenatal masculinization comes from a comparison of the auditory systems of heterosexuals and homosexuals in which click-evoked otoacoustic emissions of lesbians resembled the male pattern rather than the female pattern (McFadden, & Pasanen, 1998). Since this is a physiological difference rather than a psychological one and the sex difference appears in infancy (and from studies of opposite sex twins, appears to be influenced by the prenatal hormonal environment) this provides strong evidence that lesbianism is caused by a prenatal masculinization.

The explanation for the survival of lesbianism in spite of its apparent disadvantage could be similar to that for male homosexuals. Certain factors, environmental and genetic, lead the brain to develop more in a masculine direction. If development is shifted sufficiently far, the circuits that deal with mate choice take on a masculine character, and the female is sexually attracted to females. However, in the majority of females these genes and environmental factors merely serve to make the female personality and interests more masculine, and this frequently contributes to the female's reproductive success. One may speculate that the greater reproductive success of those with masculinity producing genes is through greater success at mate acquisition. An example would be when a tomboy is later better able to acquire a mate, because of her ability to talk about sports, and other masculine topics (in Africa, hunting perhaps).

However, in many cases the advantage of a more masculine personality may not be in mate acquisition, but in being more effective in asserting herself and hence at acquiring the resources needed for her and her children. For instance, while the very feminine female may be most attractive to a male, one who is a little more tough-minded is better at competing with another wife or mistress for the resources he is able to provide. Depending on the environment, the optimal level of masculine traits, such as assertiveness varies.

Thus, for lesbians as for male homosexuals, an occasional sexual orientation that is not conducive to reproductive success is the price paid for having mechanisms in place that provide optimal variability in personality.

So far the empirical research seems inconsistent with there being one factor that affects both lesbianism and homosexuality. While there is evidence that a gene in the Xq28 region affects homosexuality, it appears not to affect lesbianism (Hu et al. 1995). The familial studies that have shown lesbianism or homosexuality to run in families have not

found evidence that certain families have both conditions. However, it is not known if people have looked for evidence of unusually low rates of lesbianism in families with homosexuality.

Incidentally, while the evidence is not very strong, the lack of evidence of a single gene affecting the rates of both lesbianism and homosexuality is weak evidence that masculinity and femininity may have different causes.

### The Value of Male Variability

The above argument shows why a high degree of variability can contribute to male reproductive success. This provides the answer to a question that may have occurred to someone examining the earlier argument. Why is there the wide variability along the femininity-masculinity axis that creates the problem of some individuals being so far to the feminine side as to become homosexuals?

Why are not human males designed to produce the single optimum phenotype? Presumably, if the distribution shown was narrow enough, no individuals would fall into the range that produced homosexuals. Then there could be none that were so deficient in "sensitivity" etc. so as to be at a severe disadvantage in attracting and retaining females.

The reason for the wide variability in phenotypes is that long run reproductive success is facilitated by variability (see Miller 1997a for the argument in detail). Part of the reason is that reproductive success is the geometric average of reproductive success over many generations, not the arithmetic average, and the geometric average is normally below the arithmetic average. This mathematical fact implies that the more variable reproductive success is across generations the slower the population growth will be, and that there will be selection for reduced variability across generations. One way to achieve this is to have within each generation diversity in phenotypes so that at least one phenotype will be well adapted for whatever conditions occur.

Thus humans have evolved to be different from each other. This is probably achieved by differences in the prenatal environment at some stage of development. An unfortunate (for reproductive success) implication of this overall desirable process exists. It occasionally produces a homosexual male who is not attracted to females, and hence leaves few descendants.

There is evidence (Hamer et al. 1993, Hamer & Copeland, 1994, Hu et al. 1995) that a gene that promotes homosexuality has a Xq28 location, i.e. one that is on a sex chromosome. The theory about optimal diversification makes a sex location for a such a gene more plausible. The need is to diversify the behavior of sons. A location of the key gene on the X chromosome insures that there will be only one copy in sons (daughters have two X's, of course).

Imagine such a gene was on an autosomal location (i.e. not on a sex chromosome). Every so often a son would receive a double dose. If this allele had a very high probability of producing homosexuality in a double dose (a likely effect if a single dose often led to homosexuality), the combined effect of double doses producing homosexuality and single doses producing homosexuality when combined with other genes could make such an allele non-viable in the long run.

The same gene on the X chromosome is protected from producing a double dose in males. Why? Those with a double dose are daughters (by the genetic definition of female, XX is female).

There is an additional factor. A gene on the X chromosome spends twice as much time in females as males. A positive effect on the reproductive success in females could counterbalance the disadvantage of occasionally producing homosexuality in males. A gene that contributed to "femininity" could be an asset in females. With genes occasionally shifting the chromosomes they are on, one would expect a gene that was prone to contribute to homosexuality to end up on the X chromosome. This would be especially likely if the gene was sufficiently "powerful" that a double dose in males had a very high probability of producing homosexuality (and a gene which had a sufficiently powerful effect to produce such an effect in a heterozygote might plausibly produce an even stronger effect in homozygotes). An allele powerful enough to be detected by the method used by Hamer could possibly survive only if a location of the X chromosome protected males from ever inheriting a double dose.

### Frequency of Homosexuality versus Lesbianism

The theory of this paper may explain why male homosexuals are more common than female lesbians (McKnight 1997, p. 7; Wellings, Field, Johnson, & Wadsworth, 1994, p. 188-190; Sundet, Kvaem, Magnus, & Bakketeig 1988). It is a commonplace of evolutionary psychology (Symons 1990) that a male who is unusually successful in reproduction will have more grandchildren than an unusually successful female. (Unusually successful may be thought of as being in the top 10% of their sex). This is because the capacity of the female womb is limited, while a male can fertilize many women. This makes risk taking more productive for males than for females. It also implies that it pays for parents to take greater risks with the personalities of their male offspring, and to produce a wider variety of male personalities than of female personalities. Those that are not interested in the details of this argument may want to skip to the next section (Number of Older Brothers and Homosexuality) here.

Consider the benefits of producing the hyper-masculine male offspring. If the environment (including its social aspects) is of the type where such a son is most likely to be reproductively successful (sometimes through multiple wives and

mistresses), such an individual will leave many descendants. The cost of having such a hyper-masculine offspring is that if the environment is of the opposite type, the number of grandchildren will be reduced. However, there is a limit to the shortfall, since the number of offspring cannot fall below zero. Thus, the gap between the reproductive failure's offspring number, and that of the average male cannot exceed the average male's offspring number. Because a successful male can leave many offspring, while the shortfall from an unsuccessful one is limited, there are often gains from the apparently high risk strategy of producing males that are well adapted for one environment, but a failure in another. This calls for producing sons with a wide range of personalities. In particular, this implies producing son's that vary on the masculinity-femininity continuum, and a wide variability can be desirable even if it increases the risk of homosexuality.

As an illustration, imagine there are several possible types of sons. An example is given in Table 1 below. There are three types of environments and three types of sons. One type (A) has a specialized personality and be unusually empathetic and understanding. In the right social environment these traits translate into political skills and he rises to leadership of the band, and has two wives each with four children (for a total of eight). However, in the wrong environment, he is taken advantage of, and leaves no children. Another type (C) is the opposite, and has a hyper-masculine personality. In the opposite type of social environment, the hyper-masculine son is the successful one, leaving eight descendants with the empathetic, feminine one having no offspring.

The third type, B is best suited to average conditions, where he produces 4 offspring, while in the other conditions he produces 2. The example has been constructed so that there are 10 children born in each type of environment (with the only difference being which males father the offspring). For simplicity, imagine the three environments are equally probable. The two extreme personality types produce 11 offspring averaged over the three environments, while the more average type produces only 8. Selection is for the genes that make for male variability here. In the above example, producing half sons of type A and half of type C produces 10 offspring in any environment and clearly win out over producing all B's (which are best adapted for average environments).

Incidentally, a line that produced only A's or only C's would probably go extinct eventually when it hit environments its offspring were ill suited for (type A in environment c, or type C in environment a), but one that produced highly diverse offspring would stay in competition (see Miller 1997a for an elaboration of this point). In addition, parents who produce sons with both extreme types of personality have hedged their bets, and in the long run do best.

Table 1

		Environments		
		a	b	c
Son	A	0	3	8
Types	B	2	4	2
	C	8	3	0

Now consider daughters. Table 2 shows a possible pattern. The biological limit here is taken to be five offspring born (fewer surviving) and this is achieved only for the daughters who are well adapted to the environment.

Table 2

		Environments		
		a	b	c
Daughter	A	2	4	5
Types	B	4	5	4
	C	5	4	2

In the most common environment, the unspecialized type B leaves five surviving children. In the less favorable conditions she leaves four. The low variance is because she is near her capacity in all environments. Now consider the advantages of having a specialized daughter who is well suited for the extreme conditions that occur from time to time, when she has five children. In average conditions she has four. If the conditions are unfavorable for her personality type, she can fall well below her biological limit, in this example leaving only two children.

Again imagine that each of the environments occurs a third of the time. Now the most successful strategy is to always produce the unspecialized daughters who, in all conditions, bear and raise offspring at near the biological limit. Summed across all three environments, such daughters have 13 children. The more specialized daughters out reproduce her if they hit it lucky and find an environment suited to their personalities, but they do not out reproduce the average type by much, because biology constrains them. However, if the conditions are poorly suited to their personalities, they have unusually poor reproductive success. Over all three environments, such specialized daughters produce only 11 offspring. The mother who produces only the single type of daughter out reproduces the ones whose daughters are more variable.

While the particular numbers are obviously only illustrative, the effect of having an upper biological limit on female reproductive success limits the payoff to producing specialized offspring who are best suited to environments that are only occasionally encountered. While in such cases a suitable son can have spectacular reproductive success (perhaps having more than one wife) as in the above example where he had 10 offspring, the similarly specialized daughter comes up against the biological limit (in this example five offspring) and does not enjoy such a benefit.

This argument is general and suggests that daughters should be less variable than sons. In the case relevant to homosexuality, the optimal variability along the masculinity-femininity continuum would be greater for males than for females. In both sexes a disadvantage to high variability is likely to be occasional homosexuality (lesbianism for females). For males this appears to be a tolerable cost of obtaining diversity in offspring. In females with a lower value for diversity, it is less often observed.

Under these circumstances, the optimal variability for daughters is small. They are well suited for the most likely conditions, and able to function adequately in the less likely conditions. The risk of having daughters that are ideal for rare conditions is not worth running since these daughters will probably do poorly in the opposite conditions (and worse than if they had just the average type personalities). This argument suggests relatively small variability for females.

The above theory for homosexuality and lesbianism proposes that they are byproducts of adaptations to increase variability along the masculinity-femininity axis. For males a high degree of variability is selected for, and one of the costs for reproductive success of this high variability is the production of the occasional homosexual. For females, there is less benefit to variability and less variability exists. Thus, less frequently are females exposed to strong enough masculinizing influences to become lesbians.

The above effect appears strong enough to more than offset the likely greater cost of homosexuality for reproductive success that arises from the male usually being the initiating party in intercourse. In many traditional societies, virtually all women marry, and do not refuse their husbands sex. A lesbian would still marry, and even if she was not attracted to her husband, would be expected to have sex and become pregnant. The major reproductive cost to being a lesbian may be that she enjoys sex with her husband less, and this makes him a little less committed to her. Also she may not take advantage of some extra marital mating opportunities (especially important if her husband proves sterile). Thus being a lesbian would impose only a minor cost on the typical women in a prehistoric society. Of course, even this minor disadvantage does result in selection for heterosexuality, and for females to be attracted to males.



However, a male homosexual would be less likely to marry, and if married would have much less sex with his wife (and perhaps none with other females). This would be expected to put male homosexuals at a substantial disadvantage and to strongly select against homosexuality.

Given that the reproductive penalties for same sex attraction appear much greater for males than for females, one might have anticipated that in equilibrium male homosexuality would be rarer than lesbianism. Yet the opposite is found.

In the theory of this paper, the greater rate of homosexuality can be explained by the advantages to males of a high variability along the masculinity-femininity axis. This is much greater than for females, as shown above.

What genetic mechanism could make for high variability? The simplest one is an allele that makes a big difference to masculinity-femininity. Perhaps it produces a much more sensitive receptor than normal. Suppose, for instance, that (in typical conditions) if a male inherits none of the masculinity producing alleles he is of type A, if he inherits a single copy he is of type B, and if he inherits two copies he is of type C. This gene is only part of a more complicated genetic system (with many other genes that have small effects). As seen above such a high degree of variability would be desirable for males. Such a powerful masculinity producing allele could survive because the variability contributes to male reproductive success.

If the allele becomes too common, there are too many of type C, and the allele is selected against. If there are too few of the allele, it is selected for. Thus, in males such an allele could be in a balanced polymorphism and could be retained. The Xq28 located gene that Hamer and coworkers have found evidence for could be an example where a single gene has large effects, one of which is homosexuality.

However, in females an allele with such a large effect would be selected against (being replaced with a series of alleles, each of which individually had smaller effects). Imagine that a series of genes, each one of which accounted for a small effect existed. Selection would have caused these to be found in such proportions that the typical female was well adapted for the typical environment.

Now imagine such a gene with a major effect tried to invade in females. It shifts the typical female towards type C, (call that allele c). This lowers such a female's reproductive success on average, and this allele would be selected against. Likewise, an allele that shifts the typical female towards type A, call that allele a, would be selected against. We can not say whether the typical female would be aa, or cc. Notice that whether the individual is aa or cc, other minor genes would have changed in frequency to make B the average phenotype. If either aa or cc was the standard form, this would be a stable situation. If aa prevailed, c could not invade, because it would shift the individual into type C, and put her at a

disadvantage. Likewise, if initially all females were cc, the allele a could not invade because it would produce individuals of type A, and such individuals would be at a reproductive disadvantage.

Notice that the equilibrium aa and cc would both be realized with the other minor genes having evolved to have frequencies such that the typical female was of type B. In such circumstances, invasion by an allele that has a large effect in either direction can not succeed.

Notice the opposite would be true for a male. Suppose the male's minor genes made him type B and he had the genotype aa. A new mutation introduces the possibility of an ac genotype, making the individual now of type C. Since type C's on average outreproduce type B, the c allele could invade. Likewise, if the typical male was of cc genotype (and still type B because the minor genes would have adjusted in frequency to make this the most common type), type a could invade since it would produce heterozygotes ac, and these would be of type A.

Thus for males, alleles with the power to produce large shifts along the masculinity-femininity continuum would be selected for, while for females these would be selected against. In general, there are expected to be more genes having a major effect in males than in females.

Expressed in terms of game theory with the choice of offspring personality viewed as a game against nature, a mixed strategy of playing randomly different personality types is more likely to be optimal for sons than for daughters.

If this theory is correct, the search for a homosexual gene with effects large enough to be detected in a small sample is more likely to be successful than the search for a gene with a similar effect in females. Since we are just at the start of the search for genes that affect sexual orientation, the above argument suggests that such genes with major effects are much more likely to be found in males than in females.

The finding of Hamer and associates (Hamer et al. 1993; Hamer & Copeland, 1994; Hu et al. 1995) of a homosexual gene, but not a lesbian gene is consistent with this argument.

### Alternative Theories for the Survival of Homosexuality

The discussion up to this point has tried to present in a clear, logical manner how genes conducive to homosexuality could have survived. However, there are other theories. These will not be discussed in detail here because a recent book length review has just been published (McKnight, 1997) which the reader can consult for references and a discussion of alternative theories.

The only published theory that resembles the theory of this paper is that of Mellen (1981). He speculates (p. 250) that "it may well have been of adaptive value for protohuman males to become a little less brutally aggressive, a little more sensitive, a little more responsive to external influences, a little more communicative-all of which would have been possible through the retention of certain psychological traits of early adolescence or through the acquisition of a few particular feminine traits."

### Number of Older Brothers and Homosexuality

So far a theory of homosexuality has been presented. The male sexual orientation somehow takes on the female pattern, probably because a series of prenatal events changes the fetal developmental pattern. This results from the summation of a number of genetic and environmental factors. Certain alleles shift the developing fetus in the female direction, tending to produce both a greater probability of homosexuality, and a more feminine, more sensitive personality.

Are there any other facts about homosexuality which might be explained within this framework? As noted the theory explains why there are more male homosexuals than there are female ones, or lesbians. There is another effect that might be explained, the birth order effect.

In men, sexual orientation correlates with the number of older siblings (Blanchard, 1997; Blanchard, & Bogaert, 1996a; Blanchard, & Bogaert, 1996b; Blanchard & Bogaert, 1998; Blanchard, & Sheridan, 1992a; Blanchard, & Sheridan, 1992b; Blanchard, & Zucker, 1994; Blanchard, Zucker, Bradley, Hume, 1995; Blanchard, Zucker, Cohen-Kettenis, Gooren, & Bailey, 1995; Blanchard, Zucker, Siegelman, Dickey & Klassen, 1998; Jones & Blanchard, 1998). Evidence shows the effect is due to the number of older brothers, rather than the number of older sisters. Each additional older brother increases the odds of homosexuality by approximately 33% (Blanchard & Bogaert, 1996b).

Without going into great detail on the studies, this effect is not just a result of larger families being more likely to produce homosexuals, or of the parents of homosexuals being older, or similar confounding factors.

A traditional multigene additive theory cannot explain such an effect because an individual's genotype is a result of random combination of genes from the parents, and does not depend on birth order. However, of course genetic information could code for a mechanism by which the child's development depended on birth order, with the mechanism being social learning, or a change in the prenatal hormonal environment.

If homosexuality was indeed promoted by certain prenatal hormonal environments (including differences in receptor distributions), the mechanism producing higher rates of homosexuality in later born sons would also produce more feminine personalities,

Interestingly, about the time that Blanchard and associates were documenting the homosexuality birth order effect, Sulloway brought out a new book (1996) showing that there were differences in career patterns of scientists depending on birth order. In the course of this work, Sulloway did a meta-analysis of the birth order and personality literature. He looked only at studies that controlled for family size and socio-economic effects. (Many of the studies had confused socio-economic class or family size effects with birth order, since higher birth orders require large families to exist at all, and families tend to be larger in the lower socio-economic classes). He showed that there were indeed birth order effects even after these effects were controlled for.

### Birth Order Effects on the Masculinity-Femininity Continuum

Much of Sulloway's personality research also can be interpreted as younger brothers adopting a more feminine life strategy. He states (p. 77) "Status-enhancing behavior is a firstborn tendency. It is also a 'male tendency'. Cooperation is a laterborn tendency and it is also a 'female' tendency."

Later in discussing political trends he states (p. 284-285), "Tough-minded individuals tend to be leaders rather than followers, stubborn rather than flexible, and moralistic rather than empathetic. . . Not surprisingly, men are more tough-minded than women. In addition to being a male propensity, tough-mindedness is a first born characteristic." It is plausible that this trait is affected by prenatal hormonal mechanisms that differ in males and females, and between homosexual males and heterosexual females.

If the mechanism is birth order related, such that the later born become more like the recent born, it would be expected that only children would choose more sex-appropriate activities than children with older like-sex siblings, and this has been found to be the case ( $t=4.99$ ,  $p<.01$ ) (Fauls & Smith, 1956). This is true even though there is "no difference between the frequency with which children with older like-sex siblings perceive the parents as preferring sex-appropriate activities for the child and the frequency with which only children perceive parents as preferring sex-appropriate activities. ( $t=1.09$ ,  $p>.05$ )."

The later finding goes against a simple learning theory in which parents of only children try harder to teach them to prefer sex-appropriate activities.

Notice this finding about birth order would be very consistent with the finding that homosexuals have a different pattern of interest when young, and a different pattern of occupational interests when adults.

Brim (1958) has classified most of the traits found to be affected by birth order in Koch's work (dealing with six year olds) as expressive versus instrumental, which is also interpreted as feminine versus masculine (instrumental). Sulloway (1996, p.77) reanalyzed Koch's data using Brim's methodology and concluded that gender and birth order had a close relationship. Overall, he concluded the influence of birth order was two thirds as large as for sex. In dyads that included a first born girl and a later born boy, the first born girl was actually the more masculine of the two. This is consistent with the postulated mechanism acting through affecting the brain's tendency to act in a male or female typical way.

A very important finding (Eaton, Chipperfield, & Singbeil, 1989) is that birth order affects the activity levels in infants and in children. The first born are more active. Since males are generally more active than females, that is consistent with being first born producing more masculine behaviors. The National Collaborative Perinatal Survey data on 7018 children showed a decline in activity level with birth order at 8 months, 3 years, and 4 years (but not for 7 years olds or for newborns). The finding in 8 month old infants (and to a lesser degree in older ones) is important because such infants are too young for socialization to explain the observed effect, suggesting a biological cause. They also found from an observational study of children aged 5 to 9 in child care centers that the first born were more active than later borns. An earlier study (Eaton & Dureski, 1986) had reported first borns to be more active when infant activity was measured by actometers attached to the arms.

Evolution frequently seizes mechanisms which have evolved originally for other purposes. There is strong evidence that many personality traits differ between males and females. The exact mechanism for this is not known, but much of the evidence points to a hormonal mechanism.

Maccoby, Doering, Jacklin, & Kraemer (1979) have shown that the levels of testosterone in umbilical cord blood varies with birth order and Jacklin, Maccoby, & Doering (1983) have shown that timidity varies with the level of hormones. Such a mechanism could be controlled by the mother's genes. Precedents exist in the animal kingdom for biological control of behavior in accordance with birth order. Birds have been shown to have the ability to vary the amount of testosterone in the egg depending on the birth order, hence influencing their behavior (Schwabl, Mock, & Gieg, 1997)

Indirect evidence for hormones varying with birth order is that testicular cancer decreases in frequency with birth order (Prener, Hsieh, Engholm, Trichopoulos, Jensen, 1992), with the most plausible explanation being differences in prenatal hormonal exposure with birth order.

### Adult Behavior and Birth Order

Sulloway (1996) and others present evidence that birth order affects adult behavior. He has powerful evidence that younger sons are more likely to support scientific revolutions. He has collected data on thousands of scientists who were involved in 42 scientific revolutions and shows birth order effects on the positions they took. For instance, his data on several hundred scientists who were involved in the Darwinian Revolution shows that birth order was very important, with the later born much more likely to be Darwinian.

He documents birth order effects in such non-scientific areas as the French Revolution, the Protestant Reformation, and the voting of US Supreme Court Justices. The tough-minded/tender minded distinction is very important. The tough-minded tend to be first born and are willing to take strong actions to further their interests, such as suppressing dissent by force. For instance, the votes to execute the king in the French Revolution could be predicted by birth order, with the first born voting for execution. His chapter on politics (pp. 284-305), shows (among other things) that votes on the US Supreme Court can be predicted (partially) from birth order.

### Socialization and Birth Order

In ascribing the birth order effects found by Sulloway to a biological mechanism, this paper is explicitly differing from Sulloway's explanation, and the other socialization explanations often offered. Sulloway attributes the differences in behavior to childhood experiences. The environment of the first and later borns are quite different, and children develop different strategies for survival depending on their environments. This is true and his account is plausible. It is especially plausible when explaining why children of different birth orders should act differently in childhood. However, what Sulloway found was differences in adult outcomes, not childhood behavior.

A major problem with his explanation is that the environments of later born adults are not very different from that of first born adults. If all that was happening was that humans were again displaying their behavioral flexibility, we would expect that the differences observed in childhood would disappear as the children left home and moved out into the world, establishing their own families.

One possible answer to this argument is that personality traits are "hardwired" so that they cannot be changed after maturation. Given what is known about brain development, and the persistence of personality traits from childhood to adulthood (most personality traits exhibited in childhood do persist into adulthood), the persistence of personality traits that differed by birth order into adulthood is plausible. Thus, a socialization explanation is possible.

Yet there remains the question of why evolution would have designed humans so that personality traits that were useful in children necessarily persisted into adulthood. Provision for changing behavior would be especially likely if the childhood environment with its sibling competition changed radically when the child grew up, left home, and no longer had to adjust to the presence of siblings. If desirable childhood behavior resulted in undesirable adult behavior, evolution would have made it easy for adults to adopt behaviors different from their childhood behaviors. Such changes in behavior with maturation clearly occur. For instance, males become more aggressive in adolescence. Presumably if the adult behavior that maximized reproductive success did not depend on birth order, any behavioral differences would be counterproductive, and at first glance, selected against. Thus, it is very likely that the selective advantage of behavior that differs by birth order persists into adulthood, although the nature of the benefits might change. Thus, we should look for a benefit to birth order effects that persists into adulthood.

Jeremy Beer and Joe Horn (undated) using adoption study data from Texas and Colorado, compared first-born children in families with adopted children reared either as a social senior with a younger sibling or as a social junior with an older sibling (thus making it possible to control for biological order of birth effects). The only rearing order that was statistically significant was for conscientious. None of the other expected personality differences showed up. One commentator, (Cohen 1999, p. 28) notes "The clear implication is that the birth order effect is uterine rather than social, physiological rather than familial."

### Diversification by Birth Order

The author (Miller 1997a) has shown that diversification pays in evolution as well as in the business world, and genes that make two siblings pursue different life strategies are sometimes to the advantage of the parents even if not to the individuals. Perhaps the simplest example is where there are major advantages to dispersal for the survival of the parent's genes, but yet it is in the interest of both siblings to stay and to compete with their brother for the niche that the parent occupies and will vacate as they age.

An example is band leadership. If two brothers both bid for leadership, they may neutralize each others efforts and neither achieve leadership. Having one brother with a personality suited to a bid for leadership, and the other with a more cooperative personality, inclined to support the first brother in his bid is desirable. It would be in the parent's genetic interest to have a mechanism such that offspring differed in personality and followed different strategies.

A difference in dispersal strategies may also be desirable. The openness to experience that Sulloway found may serve the purpose of giving the older brothers the flexibility to concede the parental niche to a less flexible elder brother, and to seek their fortunes elsewhere, in a different environment.

One way to insure diversification is to have some mechanism is that keeps track of the number of older brothers and adjusts the personalities of the younger brothers, thus making it more likely that older and younger brothers have different personalities. The benefits of such diversification extend past childhood into adulthood. Such a mechanism could easily explain why natural selection has not limited birth order differences in behavior to childhood, but has instead produced the variations in adult behavior found by Sulloway.

### Mechanisms for the Birth Order Effects

What could be the mechanisms that produce the birth order effects?

Blanchard & Bogaert (1996b) and Blanchard & Klassen (1997) have presented a fascinating biological hypothesis for the birth order effect in homosexuality.

It is hypothesized that this fraternal birth order effect reflects the progressive immunization of some mothers to Y-linked minor histocompatibility antigens (H-Y antigen) by each succeeding male fetus, and the concomitantly increasing effects of H-Y antibodies on the sexual differentiation of the brain in each succeeding male fetus.

At first glance, if such a mechanism appeared, it would seem that it would be subject to strong selection that caused it to disappear. The mechanism seems to require both that the mother's body keep track of the number of sons she had borne and take actions that made later born ones more likely to be homosexual. Any mutation that disrupted this mechanism in the mother and prevented her from developing such progressive immunization would be expected to reduce the number of homosexual sons she bears. Any such birth order effect destroying mutation would thus be strongly selected for, unless there were strong benefits arising from keeping the mechanism intact.

Thus, the Blanchard & Klassen mechanism (and any similar mechanism by which mothers kept count of the number of sons borne and then made the later born sons more likely to be homosexual) would be easily disrupted by natural



selection. It is a multi-step process where any mutation disrupting one step would be selected for. Thus, it become critical to ask how a mechanism such as they propose could survive. If the mechanism could survive, it is at least a plausible mechanism for producing a birth order effect.

Fortunately, the above discussion of why a birth order effect might contribute to diversification explains how an effect such as Blanchard & Klaasen propose could survive. If homosexuality is merely a byproduct of a mechanism that adjusts personality in a more feminine, more sensitive direction (possibly through pre-natal hormonal effects), and there are advantages to younger sons being more feminine and less aggressively masculine, a mechanism that produces such a femininity shift could indeed survive, and even be selected for. A younger son being more sensitive and flexible facilitates his competing for resources during childhood. In adulthood, it avoids competition of the younger brothers with the older ones.

There is scope for parent-offspring conflict regarding diversification. Each son maximizes his reproductive success if he competes for the most desirable niche, perhaps a leadership position. The parental reproductive interest is in diversification, and this may mean having one son (the first?) take the most attractive niche, and then others compete for other niches. A mechanism in the mother that moderated the younger sons' competitive drive could be in the mother's reproductive interest. The Blanchard & Klassen H-Y antigen mechanism might do this through somehow changing the "masculinity" of the later sons. Such a mechanism coded into the mother's genes could be selected for.

However, such selection not to compete may not be in the interest of the later born sons themselves. Although they share half of their genes with their brothers, their reproductive interest is still in competing for the niches that would give them the greatest number of offspring. The sons might evolve countermeasures to the mother's attempts to direct their development.

Any mechanism in the son (say a change in the blood/brain barrier) that kept the mother's antibodies from affecting her son's brain and increasing its probability of being homosexual would be selected for. It would lower the probability of that son being homosexual, and hence increase the number of descendants he would have.

If the effect on the later born sons was to shift their personalities in a more feminine direction, the personality of these sons would be too feminine and not aggressive enough. Suppose the evolution of these measures in the mothers had not changed the personalities of the elder sons. This implies that averaged over all sons, the personalities would be too feminine, too flexible. The average male would not be "masculine" enough for his own maximal reproductive success. This

would produce a general selection in males for a more aggressive, more masculine personality, which is then moderated by the mother's influence back towards the more optimal level. Selection for male masculinity genes stops when the average male (average of first and later born ones) has the optimal genotype for these traits. Since the probability of a particular gene being passed on is the same for first and later borns, the selection acts on the average male (some first born, some later born). When the average male has the optimal genotype, the first born males will be too masculine, too aggressive. The later born males are shifted in a more feminine direction, with a possibility that the last born are too feminine for maximal reproductive success.

Note how in equilibrium, average masculinity is probably unchanged from what would exist in the absence of the maternal mechanism which adjusts masculinity to birth order. The first born males are more masculine and less likely to be homosexual than they would if this maternal mechanism did not exist. The later born are more likely to be homosexuals, but the average rate of homosexuality is probably unchanged.

One's first reaction on hearing of a mechanism that might make later borns sons more likely to be homosexual is that the resulting increase in homosexuality would reduce the both the mother and the sons reproductive success. It would hence be strongly selected against. The above argument shows that this need not be the effect in full equilibrium.

A maternal mechanism that operates on the degree of "masculinity" can explain the homosexuality birth order effect. The homosexuality is just a byproduct of shifting in the feminine direction.

It is possible that some birth order effects are produced by other than a hormonal mechanism, or possibly through socialization. As Sulloway points out, not all birth order effects are parallel to gender. Conscientiousness is strongest in the first born. His literature review (p. 74) found 20 studies showing this result and none showing the opposite. Yet, females are usually more conscientious than males. The explanation may be social with parents devoting more effort to inculcating conscientiousness in their first born (especially when they are young and the parents can devote their full efforts to the child), or as Sulloway speculates it could be related to the first born seeking parental approval by conforming to parental norms. Interestingly, conscientiousness (Beer and Horn, undated) is the one personality trait for which there is evidence that the social effects of having siblings can produce a "birth order" effect.

There may be an evolutionary advantage to first borns being unusually conscientious. Very often the older siblings help take care of the younger siblings. Doing so successfully requires a high degree of conscientiousness. Since the younger sibling are carrying many of the same genes as the older siblings, genes that made for conscientiousness in older siblings

would be selected for. Given the low cognitive abilities of most children, rigidly following of parental rules would make them better care takers for even younger children than a more flexible personality. Since conscientiousness usually increases with age, to achieve the level of conscientiousness in a child that would make him or her a good care-giver for the younger siblings (or at least would keep them from using their greater size to harm the younger ones) would require the sort of design that would make for a highly conscientious, perhaps even rigid adult. The inflexibility and toughness in first born adults Sulloway found may be a result of a mechanism designed to insure they rigidly follow parental instructions when young.

Younger siblings would not benefit as much from conscientiousness. They less often take care of even younger siblings. Because they are usually smaller and weaker than the older siblings, they do not need conscientiousness and a tendency to obey rules to avoid hurting the other siblings. Instead openness to experience and flexibility may be required to obtain the resources needed for them to survive childhood.

In considering the evolutionary probabilities, it should be noted that high conscientiousness in the first born (and other early born) is more strongly in the mother's interest (her genes benefit from having the younger children cared for and not hurt) than in the interest of the first born itself (who may be called on to sacrifice their immediate interest to care for the younger ones, or to avoid hurting them). Mothers would be strongly selected for a mechanism which kept track of birth order, and somehow modified the personalities of the offspring to achieve the optimal combination.

The beauty of the mechanism proposed by Blanchard & Klassen is that it provides a way for the mother to keep track of the number of sons borne and to have the personalities differ. This insures the desired diversification.

The above mechanism could also explain why boys with gender identity disorder had a different sibling sex ratio (Zucker, Green, Coates, Zuger, Cohen-Kettenis, Zecca, Lertora, Money, Hahn-Burke, Bradley, & Blanchard, 1997) since those with older brothers would have more feminizing effects. The same set of events could also cause certain boys to have the parts of their brain that related to gender identity feminized.

Having behavior depend on birth order could be achieved by other mechanisms. Such mechanisms would require that somehow the mother's body retain knowledge how many previous children had been born and their sex. There is one other mechanism known which could retain this information. Male fetal progenitor cells persist in maternal blood for as long as 27 years postpartum (Bianchi, Zickwolf, Weil, & Sylvester, 1996). It is possible that the mother might somehow use this information to modify the personality of her sons, thus achieving optimal diversification.

## Conclusion

The survival of a human predisposition for homosexuality can be explained by sexual orientation being a trait that is influenced by a number of pleiotropic genes. These operate during the development process to shift male development, including that of the brain, in the female direction. If by chance, a male inherits numerous alleles that shift his development in the feminine direction he becomes homosexual or bisexual. However, more often these alleles are inherited along with other genes that make the male heterosexual. Then the effect of the alleles is to make for greater sensitivity, empathy, tendermindedness, and kindness.

These traits make heterosexual carriers of the genes better fathers and more attractive mates. As long as such alleles sometimes lead to homosexuality, evolution will act so as to increase the frequency of other alleles that produce males who are too masculine for maximal reproductive success. This creates a balanced polymorphism in which the feminizing effect of these alleles in heterosexuals offsets the adverse effects (on reproductive success) of these alleles contribution to homosexuality. A similar effect probably occurs for genes that can produce lesbianism in females.

The whole system survives because it serves to provide a high degree of variability among the personalities of offspring, providing the genotype with diversification, and reducing competition among offspring for the same niches. Because extremes of personality are more advantageous to males, the variability along the masculine/femininity axis is greater for males. This is reflected in male homosexuals being more common than female ones.

The birth order effect on homosexuality is probably a byproduct of a mechanism that shifts personalities more in the feminine direction in the later born sons, reducing the probability of these sons engaging in unproductive competition with each other.

The above has implications for sociobiology. The requirement that a causal mechanism survive over evolutionary time imposes constraints on the set of possible mechanisms to explain homosexuality. A mechanism (i.e. a gene for homosexuality) which only produces homosexuality would be strongly selected against and could not survive. A pleiotropic gene for which homosexuality was one of only several mechanisms could survive. The evolutionary perspective contributes to understanding the causes of a puzzling phenomenon.

## References

- Allen, L. S. and Gorski, R. A. (1992). Sexual orientation and the size of the anterior commissure in the human brain. Proc Natl Acad Sci USA. 89: 7199-7202.
- Alexander, J. E. and Sufka, K. J. (1993). Cerebral lateralization in homosexual males? A preliminary EEG investigation. Intl J Psychophysiol. 15: 269-274.
- Bailey, J. M., Finkel, E., Blackwelder, K. and Bailey, T. (1996). Masculinity, femininity, and sexual orientation. (unpublished manuscript).
- Bailey, J. M., Martin, N. G. (1995). A twin registry study of sexual orientation, poster presentation at the International Academy of Sex Research, 21st Annual Meeting, Provincetown, Massachusetts.
- Bailey, J. M., Kim, P., Hills, A. and Linsenmeier, J. Butch, femme, or straight-acting? Partner preferences of gay men and lesbians, undated.
- Bailey, J. M., Pillard, R.C. (1991). A Genetic Study of Male Sexual Orientation. Arch Gen Psychiatry. 1089-1096.
- Bailey, J. M., Pillard, R. C., Neale, M. C., Agyei, Y. (1993). Heritable factors influence sexual orientation in women. Arch Gen Psychiatry. 217-223.
- Bailey, J. M., and Zucker, K. J. (1995). Childhood sex-type behavior and sexual orientation: A conceptual analysis and quantitative review. Dev Psychol. 31: 43-55.
- Baker, R. and Bellis, M. Human Sperm Competition. London: Chapman and Hall, 1995.
- Bakker, J. and Slob, A. K., (1997). Sexual differentiation of the brain and partner preferences in the male rat: Behavioral, neuroanatomical and neuroimmunocytochemical studies. In Sexual Orientation : Toward Biological Understanding (eds: L. Ellis and L. Ebertz), 91-106.
- Beer, J. and Horn, J. M. (undated). The influence of rearing order on personality development within two adoption cohorts. Manuscript in review. University of Texas at Austin.
- Bell, A. P., and Weinberg, M. (1978). Homosexualities: A study of diversity among men and women. New York: Simon and Schuster.
- Bell, A. P., Weinberg, M. and Hammersmith, S. (1981). Sexual Preference: Its Development in Men and Women. Bloomington: Indiana University Press.
- Bianchi, D. W., Zickwolf, G. K., Weil, G. J., and Sylvester, S. (1996). Male Fetal Progenitor Cells persist in Maternal Blood for as long as 27 years Postpartum, Proc Natl Acad Sci USA. Vol. 93: 705-708.

- Blanchard, R.(1997). Birth order and sibling sex ratio in homosexual versus heterosexual males and females. Annual Review of Sex Research, Volume 8, 27-67.
- Blanchard, R., and Bogaert, A. F. (1996a). Biodemographic comparisons of homosexual and heterosexual men in the Kinsey interview data. Am J Psychiatry. 153: 27-31.
- Blanchard, R., and Bogaert, A. F. (1996b). Homosexuality in men and number of older brothers. Arch Sex Behav. 25: 545-573.
- Blanchard, R., and Bogaert, A. F. (1998). Birth order in homosexual versus heterosexual sex offenders against children, pubescents, and adults. Arch Sex Behav. 27:595-603.
- Blanchard, R., and Klassen, P. (1997). H-Y antigen and homosexuality in men. J Theor Biol. 185: 373-378.
- Blanchard, R., and Sheridan, P. (1992a). Proportion of unmarried siblings of homosexual and nonhomosexual gender-dysphoric patients. Can J Psychiatry.37: 163-167.
- Blanchard, R., and Sheridan, P. (1992b). Sibship size, sibling sex ratio, birth order, and parental age in homosexual and nonhomosexual gender dysphorics. J Nerv Ment Dis.180: 40-47.
- Blanchard, R., and Zucker, K. (1994). Reanalysis of Bell, Weinberg, and Hammersmith's data on birth order, sibling sex ratio, and parental age in homosexual men. Am J Psychiatry. 151: 1375-1376.
- Blanchard, R., Zucker, K., Bradley, S. J., Hume, C. (1995). Birth order and sibling sex ratio in homosexual male adolescents and probably prehomosexual feminine boys. Dev Psychol. 31: 22-30.
- Blanchard, R., Zucker, Cohen-Kettenis, P., Gooren, L., and Bailey, J. M. (1995). Birth order and sibling sex ratio in two samples of Dutch gender-dysphoric homosexual males. Arch Sex Behav. 25: 495-514.
- Blanchard, R., Zucker, K., Siegelman, M., Dickey, R., and Klassen, P., (1998). The relation of birth order to sexual orientation in men and women. J Biosoc Sci .30:511-519.
- Bogaert, A. F. and Blanchard, R. (1996). Physical development and sexual orientation in men: Height, weight, and age of puberty differences. *Personality and Individual Differences*, 21: 77-84.
- Brim, Orville G. (1958). Family structure and sex role learning by children: A further analysis of Helen Koch's data. Sociometry, 21: 1-16.
- Buss, D. M. (1989). The Evolution of Desire. NY: Basic Books.

- Buss, D. M. (1994). Sex differences in human mate preference: Evolutionary hypotheses tested in 37 cultures. Behav Brain Sci. 12: 1-49, 1989.
- Buss, D. M. and Barnes, M. (1994). Preferences in human mate selection. J Pers Soc Psychol. 50: 559-570.
- Chung, Y. B. and Harmon, L. W. (1994). The career interests and aspirations of gay men: How sex role orientation is related. J of Vocational Behav. 45: 223-239.
- Cohen, David B. (1999). Stranger in the Nest: Do Parents Really Shape Their Child's Personality, Intelligence, or Character? New York: John Wiley & Sons (p. 28).
- Dorner, G., Rohde W., Stahl, F. et al. (1975). A neuroendocrine predisposition for homosexuality in men. Arch Sex Behav. 4: 1-8.
- Dorner, G., Schenck, B., Shmiedel, B and Ahrens L. (1983). Stressful events in prenatal life of bi and homosexual men. Exp Clin endocrinol Diabetes. 81: 83-87.
- Eaton, W., Chipperfield, J. and Singbeil, C. (1989). Birth order and activity level in children. Dev Psychol. 25: 668-672.
- Eaton, W., and Dureski, C. M. (1986). Parent and actometer measures of motor activity in the young infant. Infant Behavior and Development, 9: 383-393.
- Eaves, L. J., Eysenck, H. J. and Martin, N. G. (1989). Genes, Culture, and Personality. London: Academic Press.
- Ellis, L.(1997). Perinatal Influences on Behavior and Health, with Special Emphasis on Sexual Orientation and Other Sex-Linked Behavior. Sexual Orientation : Toward Biological Understanding (eds: L. Ellis and L. Ebertz), 71-88.
- Ellis, L., and Ames, M. A. (1987). Neurohormonal functioning and sexual orientation: a theory of homosexuality-heterosexuality. Psychol Bull. 101: 233-258.
- Ellis, L., Ames, M. A., Peckham, W. and Burke, D. (1987). Sexual orientation of human offspring may be altered by severe maternal stress during pregnancy. The J of Sex Research, 25: 152-157.
- Ellis, L. and Ebertz, L. (eds) (1997). Sexual Orientation : Toward Biological Understanding, (eds: L. Ellis and L. Ebertz).
- Ellis, L., Hoffman, H., and Burke, D. M. (1990). Sex, sexual orientation and criminal and violent behavior. Personality and Individual Differences, 11: 1207-1212.
- Eysenck, H. J. and Wilson, G. (1979). The Psychology of Sex. London: J. M. Dent and Sons.
- Fauls, L. B. and Smith, W. D. (1956). Sex role learning of five-year-olds. J Genet Psychol. 89: 105-117.

- Gallup, G. and Suarez, S. (1983). Homosexuality as a by-product of selection for optimal heterosexual strategies. Perspect Biol Med. 26: 315-322.
- Gandelman, R. (1992). Psychobiology of behavioral development. Oxford: Oxford University Press.
- Getz, W. (1993). Invasion and maintenance of alleles that influence reproductive success. J Theor Biol. 162: 515-537.
- Gettelman, T. E. and Thomas, J. K. (1993). Actual differences and stereotypical perceptions in body image and eating disturbance: A comparison of male and female heterosexual and homosexual samples. Sex Roles. 29: 545-562.
- Gladue, B. A., Beatty, W. W., Larson, J. and Staton, R. D.,(1990). Sexual orientation and spatial ability in men and women. Psychobiology. 18(1): 101-108.
- Gladue, B. A., Green, R., and Hellman, R. E. (1984). Neuroendocrine response to estrogen and sexual orientation. Psychoneuroendocrinology. 20: 475-485.
- Goode, E. and Troiden, R. R. (February,1980). Correlates and Accompaniments of Promiscuous Sex Among Male Homosexuals. Psychiatry. 43: 51-59.
- Gooren, L. J. G. (1986). The neuroendocrine response of luteinizing hormone to estrogen administration in heterosexual, homosexual, and transsexual subjects. J Clin Endocrinol Metab. 63: 588-593.
- Gorman, Michael R. (1994). Male homosexual desire: Neurological investigations and scientific bias. Perspect Biol Med. 38(1).
- Green, R. (1987). The "Sissy Boy Syndrome" and the Development of Homosexuality, New Haven: Yale University Press.
- Guadio, R. P. (1994). Sounding gay: Pitch properties in the speech of gay and straight men. American Speech. V 69 N 1.
- Hall, J. A. Y. and Kimura, D. (1994). Dermatoglyphic asymmetry and sexual orientation in men. Behav Neurosci. 108: 1203-1206.
- Hall, J. A. Y. and Kimura, D. (1995). Sexual orientation and performance on sexually dimorphic motor tasks. Arch Sex Behav. 24(4): 395-407.
- Halpern, D. F. and Crothers, M. (1997). Sex, sexual orientation, and cognition, Sexual Orientation : Toward Biological Understanding (eds: Lee Ellis and Linda Ebertz),181-197.
- Hamer, D and Copeland, P. (1994). The Science of Desire. New York: Simon and Schuster.
- Hamer, D and Copeland, P. (1998). Living with our Genes. New York: Doubleday.

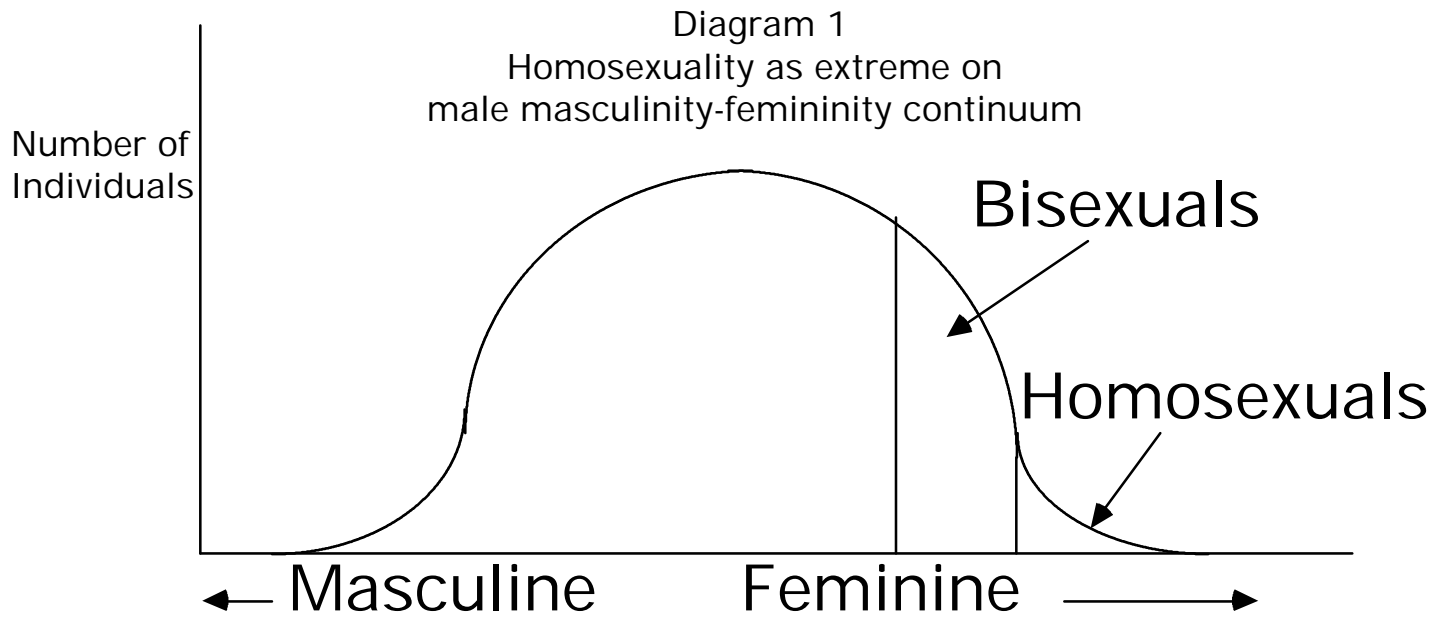


- Hamer, D and Hu, S., Magnuson, V., Hu, N. and Pattatuci, A. (1993). A linkage between DNA markers on the X chromosome and male sexual orientation. Science. 261: 321-327.
- Hu, S., Pattatuci, A., Patterson, C., Li, L., Fulker, D. W., Cherny, S., Kruglyak L, and Hamer, D .(1995). Linkage between sexual orientation and chromosome Xq28 in males but not in females. Nat Genet. 11: 248-256.
- Jacklin, C. N., Maccoby, E., and Doering, C. H. (1983). Neonatal sex-steroid hormones and timidity in 6-18-month-old boys and girls, Dev Psychobiol. 16(3): 163-168.
- Jones, M. B. & Blanchard, R., (1998). Birth order and male homosexuality: Extension of Slater's index., Hum Biol. 70:775-787.
- Kallmann, F. J. (1952). Comparative twin studies on the genetic aspects of male homosexuality. J Nerv Ment Dis. 115:283-298.
- Laumann, E., Gagnon, J. H., Michael R. T., Michaels, S. (1994). The Social Organization of Sexuality: Sexual Practices in the United States. Chicago: University of Chicago Press.
- LeVay, S. (1991). A difference in hypothalamic structure between heterosexual and homosexual men. Science. 253: 1034-1037.
- LeVay, S. (1993). The Sexual Brain. Cambridge, MIT Press.
- LeVay, S. (1996). Queer Science: The Use and Abuse of Research into Homosexuality. Cambridge, MIT Press.
- MacIntyre, F. and Estep, K. W. (1993). Sperm competition and the persistence of the genes for male homosexuality. Biosystems. 31: 223-233.
- Maccoby, E. E., Doering, C. H., Jacklin, C. N., and Kraemer, H. (1979). Concentrations of sex hormones in umbilical cord blood: Their relation to sex and birth order in infants. Child Dev. 50: 632-642.
- McCormick, C. M., and Witelson, S. F. (1991). A cognitive profile of homosexual men compared to heterosexual men and women. Psychoneuroendocrinology. 16: 459-473.
- McFadden, D. and Pasanen, E. G. (1998). Comparison of the auditory systems of heterosexuals and homosexuals: Click-evoked otoacoustic emissions. Proc Natl Acad Sci USA. 95, 2709-2713.
- McGuire, T. R. (1995). Is homosexuality genetic? A critical review and some suggestions. J Homosex. 28(1/2): 115-144.
- McKnight, J. (1997). Straight Science: Homosexuality, Evolution and Adaptation. New York: Routledge.
- Mellen, S. L. (1981). The Evolution of Love. San Francisco, Freeman.

- Miller, E. M. (1994). Prenatal sex hormone transfer: A reason to study opposite-sex twins, Personality and Individual Differences, 17, 511-529.
- Miller, E. M. (1997a). Could nonshared environmental variance have evolved to assure diversification through randomness? Evolution and Human Behavior, 18, 195-221.
- Miller, E. M. (1997b). Evidence from opposite-sex twins for the effects of prenatal sex hormones. in Males, Females, and Behavior: Towards Biological Understanding, Lee Ellis & Linda Ebertz, Eds. Westport, CT: Praeger.
- Moran, P. A. P. (1972). Familial effects in schizophrenia and homosexuality. Aust NZ J Psychiatry, 6: 116-119.
- Murray, S. O. (1991). Homosexual occupations in Meso-America. J Homosex, 21: 57-65.
- Perkins, M. W. (1981). Female homosexuality and body build. Arch Sex Behav, 10: 337-345.
- Pillard, R. C. (1991). Masculinity and femininity in homosexuality: "Inversion" revisited. In J. C. Gonsiorek and Weinrich, J. D. (Eds.). Homosexuality: Research Implications for Public Policy, (pp. 32-43). Newbury Park, Ct: Sage.
- Pillard, R. C., and Bailey, J. M. (1998). Human sexual orientation has a heritable component. Hum Biol, 347-365.
- Pillard, R.C., and Weinrich, J.D. (1986). Evidence of familial nature of male homosexuality. Arch Gen Psychiatry, 43:808-12.
- Prener, A , Hsieh, C., Engholm, G. , Trichopoulos, D., Jensen, O. (1992). Birth order and risk of testicular cancer. Cancer Causes Control, 3: 265 - 272.
- Plomin, R., Chipuer, H. M., and Neiderhiser, J. M. (1994). Behavior genetic evidence for the importance of nonshared environment. In Hetherington, E. M., Reiss, D., and Plomin, R. Eds. Separate Social Worlds of Siblings: The Impact of Nonshared Environment on Development. Hillsdale: Lawrence Erlbaum.
- Reite, M., Scheeder, J., Richardson, D., and Teale, P. (1995). Cerebral laterality in homosexual males: Preliminary communication using magnetoencephalography. Arch Sex Behav, 24: 585-593.
- Rogers, S. M., and Turner, C. F. (1991). Male-male sexual contact in the USA: Findings from five sample surveys, 1970-1990, J of Sex Research, 28 (4):491-519.
- Rowe, D. C. (1994). The Limits of Family Influence: Genes, Experience, and Behavior. (New York: Guilford: 1994).
- Saghir, M. T. and Robins, E. (1973). Male and Female Homosexuality. Baltimore, Williams and Wilks.
- Salais, D. and Foscher, R. B. (1995). Sexual preference and altruism. J Homosex, 28, 185-196.

- Schlegel, W. S. Die Sexualinstinkte de Menschen, Munich: Tutten Verlag.
- Siever, M. D. (1994). Sexual orientation and gender as factors in socioculturally acquired vulnerability to body dissatisfaction and eating disorders. *J of Consulting and Clinical Psychology*, 62: 252-260.
- Spence, J. T. and Helmreich, R. L. (1978). Masculinity and Femininity. Austin, University of Texas Press.
- Sprecher, S. (1989). The importance to males and females of physical attractiveness, earnings potential, and expressiveness in initial attraction. *Sex Roles*, 21, 591-605.
- Sprecher, S., Sullivan, Q and Hartfield, E. (1994). Mate selection preferences: Gender differences examined in a national sample. *J Per Soc Psychol*, 66: 1074-1080.
- Sulloway, Frank J. (1996). Born to Rebel: Birth Order, Family Dynamics, and Creative Lives. New York: Pantheon Books.
- Sundet, J. M., I. L. Kvale, P. Magnus, and L.S. Bakketeig (1988). Prevalence of risk-prone behavior in the central population of Norway, in A. F. Fleming, M. Carballo, and D. F Fitzsimons (eds), The Global Impact of Aids. London: Alan R. Liss.
- Swaab, D. F. and Hofman, M. A. (1990). An enlarged suprachiasmatic nucleus in homosexual men. *Brain Research*, 537: 141-148.
- Swaab, D. F. and Hofman, M. A. (1995). Sexual differentiation in the human hypothalamus in relation to gender and sexual orientation. [Review]. *Trends Neurosci*, 18: 264-270.
- Swaab, D. F., Slob, A. K., Houtsmuller, E. J., Brand, T. and Zhou, J. N. (1995). Increased number of vasopressin neurons in the suprachiasmatic nucleus (SCN) of "bisexual" adult male rats following perinatal treatment with the aromatase blocker ATD. *Developmental Brain Research*, 85: 273-279.
- Swaab, D. F., Zhou, J. N., Fodor, M., and Jofman, M.A. (1997). Sexual differentiation of the human hypothalamus: Differences according to sex, sexual orientation, and transsexuality. Sexual Orientation: Toward Biological Understanding (eds: Lee Ellis and Linda Ebertz), 129-150.
- Symons, D. (1990). The Evolution of Human Sexuality. New York, Oxford University Press.
- Tuttle, G. E. and Pillard, R. C. (1991). Sexual orientation and cognitive abilities. *Arch Sex Behav*, 13: 427-439.
- Van de Ven, P., Rodden, P., Crawford, J. and Kippax, S. (1997). A comparative demographic and sexual profile of older homosexually active men. *The J of Sex Research*, 34, 349-360.

- Wegesin, D. J. (1998). Event-related potentials in homosexual and heterosexual men and women: Sex-dimorphic patterns in verbal asymmetries and mental rotation. Brain Cogn. 36, 73-92.
- Weinrich, J. D. (1987). Sexual Landscapes. New York: Scribner's Sons.
- Weinrich, James D. (1976). Human Reproductive Strategies (Doctorial Dissertation Harvard University). University Microfilms no. 77-8348.
- Wellings, K., Field, J., Johnson, A., and Wadsworth, J. (1994). Sexual Behavior in Britain. Penguin Books Ltd.
- Werner, D. (1998). On the Evolution and Cross-Cultural Variation in Male Homosexuality, (English Translation of: Sobre a evolução e variaçã o cultural na homossexualidade masculina. In Joana Maria Pedro e Miriam Pillar Grossi (orgs). Masculino, Feminino Plural. Florianópolis: ed. Mulheres, pgs.99-129.
- Whitam, F. L., Diamond, M., and Martin, J. (1993). Homosexual orientation in twins : a report on 61 pairs and 3 triplet sets. Arch Sex Behav. 22:187-206.
- Wilson, E. O. (1975). Sociobiology: The New Synthesis. Cambridge, Harvard University Press.
- Zucker, K. J., Green, R., Coates, S., Zuger, B., Cohen-Kettenis, P. T., Zecca, G. M., Lertora, V., Money, J., Hahn-Burke, S., Bradley, S. J., and Blanchard, R. (1997). Sibling sex ratio of boys with gender identity disorder. J Child Psychol Psychiatry. 38: 543-551.
- Zucker, K. J., and Bradley, S. (1995). Gender Identity Disorder and Psychosexual Problems in Children and Adults. New York, Guilford Press.




---

Footnotes

<sup>1</sup> In considering how genes for homosexuality could survive, I find it very implausible that genes whose only effect was to produce homosexuality could survive. Homosexuality is just too negative for reproductive success. While in theory homosexual genes could survive if they increased the reproductive success of others carrying such genes (such as by being helpers at the nest, as is found in some birds), there is no real evidence for an effect anywhere near strong enough to insure the survival of such genes (for discussions of such theories such McKnight, 1997, or Weinrich, 1987). No one has come up with plausible reasons why homosexuality would have emerged rather than mere asexuality.

<sup>2</sup> The case where the other effects are adverse to reproductive success does not have to be considered in detail, since genes that both hindered reproduction in heterosexuals and produced homosexuality would be quickly eliminated from the gene pool.